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A Suspected Congenital Cardiac Deficiency

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tion with a continuous suture. The skin and muscles were sutured with 13 interrupted through and through sutures and the skin was finally sutured with a continuous suture. Nylon, No. 00, suture material was used for all of the above sutures.

The artificial respirator was gradually suppressed, the patient allowed to practice normal respiration which it did with no difficulty and the tracheal tube was then removed.

The patient recovered from the anesthesia seven hours later. At this time 1000 cc. of 5 percent dextrose solution was administered intravenously.

On the following morning the patient exhibited mild discomfort and 5 mg. acetyl salicylic acid were given per orum as a sedative.

On the following days the patient manifested no discomfort, respiration was normal, the wound healed uneventfully and the patient was discharged on Nov. 20, 1949.

William Fennessy, '51

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2 **Unusual Bovine Horn Studs.** On Jan. 17, 1950, the ambulatory clinicians at Iowa State College were called to see a 2-year-old Shorthorn heifer with a history of having been dehorned about 18 months previously, but horn stubs had grown out on the right side of the poll since that time.

The two horn stubs resembled miniature goat horns, being approximately 4 in. long, and ⅔ in. in diameter, and adjacent to each other. About 1 in. of the shell on the end of one of the horn stubs was broken off, thus causing continuous hemorrhage. The hair on the right side of the head was almost completely matted with blood.

It was decided to remove the horny growths and the animal was restrained in a stanchion with a nose lead. A local anesthetic of 10 cc. of 4 percent procaine hydrochloride was injected midway between the orbit and the base of the horn stubs and about ½ in. lateral to the edge of the frontal bone with a ¾ in. 16 gauge needle in an attempt to block the cornual nerve. The horn stubs along with the skin in which they were imbedded were removed with ranch-type dehorners. Hemorrhage was controlled by clamping off the larger blood vessels with hemostats.

Fig. 1. Horn stubs.

Horn stubs are a common sequel of dehorning operations due to the failure to destroy or remove all of the germinal epithelium surrounding the base of the horns.

Donald H. Crawford '50

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3 **A Suspected Congenital Cardiac Deficiency.** On Jan. 12, 1950, a 9-month-old female bovine of the Angus breed was admitted to Stange Memorial Clinic. Accompanying history stated that recently the animal showed weakness in the shoulders and had an enlargement in the area of the umbilicus. One year previously, two calves in the same herd exhibited similar symptoms. One of these two animals died; the other was slaughtered. No post mortem results on the slaughtered animal were obtained.

Clinical examination of the patient revealed slightly accelerated respirations and a normal temperature. Edema of the brisket was quite evident. The front legs and shoulders were turned outward and the animal showed considerable respira-

tory distress after exertion. Auscultation of the respiratory organs revealed no evidence of rales. The pulse was slightly accelerated, and a prominent jugular pulse was observed. On Jan. 13, 1950, a blood sample was drawn and sent to the clinical laboratory for a differential white blood cell count. Results of the count were within normal limits, indicating that a bacterial infection was not involved.

The next four days showed a progressive increase in the edema of the underline with increasing dyspnea. The patient died on Jan. 18, 1950.

The cadaver was removed to the post mortem laboratory where a necropsy examination was performed. A generalized subcutaneous edema was found. The thoracic cavity was completely filled with serous fluid. Ascites was also demonstrated by the presence of a great amount of this fluid in the peritoneal cavity. Extreme diffuse hepatic cirrhosis was observed and widespread hemorrhages were seen on the thoracic and abdominal viscera. Passive congestion was also quite pronounced on the abdominal organs. The right atrium and right ventricle of the heart had undergone extreme hypertrophy. Examination of the right atrioventricular valves revealed that complete closure was impossible because of the distortion caused by the hypertrophied condition of the atrium and ventricle. Passive congestion of the brain was also noted. Cultures taken from the lungs, spleen, liver and heart were negative for bacterial growth.

In order to satisfactorily and logically explain these lesions and the ante-mortem symptoms observed it seems necessary to start with a valvular insufficiency. With the tricuspid valves not closing properely, there resulted an accumulation of blood in the right atrium. This resulted in dilatation and subsequent hypertrophy of the right side of the heart which only helped to exaggerate the valvular insufficiency. With each systole of the right ventricle, some blood was forced in a reverse direction back into the vena cava emptying into the right atrium and caused the juglar pulse so noticeable on ante-mortem examination. This was also the cause of the passive congestion of the brain.

This back pressure in the posterior vena cava caused a passive congestion of the liver. With the engorgement and stagnation of the circulation in this organ, death of the hepatic cells resulted with subsequent proliferation of the connective tissue stroma causing the extreme cirrhotic condition we observed. This cirrhosis of the liver retarded portal circulation and resulted in the passive congestion observed in the abdominal viscera and the hydroperitoneum. Since the hepatic epithelium was displaced by connective tissue and function of the liver was impaired, less albumin was produced for the blood plasma. This decrease in serum albumin lowered the colloidal osmotic pressure of the blood which further aggravated the developing ascites, hydrothorax and anasarca.

The extreme hydrothorax condition mechanically impeded respiration and caused the dyspnea observed in the living animal. In view of the absence of bacterial infection or any other evident etiological factor, and the history stating that two other younger animals in this same herd had shown similar symptoms, it seems logical to conclude that this is a congenital cardiac condition. As growth of the animal progressed the demand on the heart increased to a point which it could no longer meet because of its valvular insufficiency. In order to compensate for this demand, hypertrophy of the heart musculature occurred, only to aggravate the insufficiency and to precipitate the above mentioned pathological changes.

R. M. Hacecky, '50

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4 **Suppurative Nephritis in a Hereford Cow.** A registered 6-year-old Hereford cow entered Stange Memorial Clinic on Dec. 30, 1949 with a history of having been ill since Nov. 21. A tentative diagnosis of traumatic gastritis had been made in the field immediately after her illness was noted. Treatment given at this